

Exhibit 1

Specific Causation Expert Report for Jefferson Criswell Thomas Longo, M.D.

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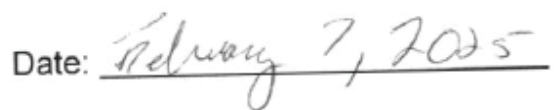
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		Chart 1: 1L	Chart 2: ATSDR	Chart 3: Deposition/FM
	Cumulative ug/l-M	Cumulative consumption (total ug= days*concentration per L)	Cumulative consumption (total ug= days*concentration per ATSDR exposure assumptions)	Cumulative consumption (total ug= days*concentration per deposition/FM exposure assumptions)
Hadnot Point				
TCE	8,357	86,022	345,628	534,241
PCE	258	2,640	23,337	34,466
VC	380	3,897	34,441	50,865
BZ	85	858	7,581	11,196
Terawa Terrace				
TCE	46	911	3,905	6,036
PCE (TechFlowMP Model)	1,147	22,510	96,472	149,118
PCE (MT3DMS Model)	1,594	31,267	134,002	207,128
VC	82	1,610	6,899	10,663
BZ	-	-	-	-
Totals HP & TT				
TCE	8,403	86,933	349,534	540,278
PCE (TechFlowMP Model)	1,405	25,151	119,809	183,584
PCE (MT3DMS Model)	1,852	33,908	157,339	241,594
VC	462	5,507	41,339	61,528
BZ	85	858	7,581	11,196

I have also reviewed the reports of Drs. Benjamin Hatten and Steven Bird, who both identified hazardous levels of exposure that demonstrate toxic levels of TCE, PCE, vinyl chloride and benzene that Mr. Criswell met or exceeded.

Viewing Dr. Reynolds' exposure numbers against these demonstrated toxic levels clearly establishes that Mr. Criswell's exposure was significant and substantial. Mr. Criswell exceeds each of the demonstrated levels set out here.

The opinions of Dr. Reynolds, Dr. Hatten, and Dr. Bird confirm that Mr. Criswell's exposure to the chemicals at Camp Lejeune has been documented in other literature to have a positive association with the diagnosis of bladder cancer.

VII. General causation

Before advancing to the application of a differential etiology for Mr. Criswell it is important to first recognize whether there is enough evidence to establish whether the

chemicals in the water at Camp Lejeune are capable of causing bladder cancer as a general matter.

Numerous regulatory and scientific bodies have recognized that these four chemicals are toxic and capable of causing cancer. IARC recognizes TCE, VC, and benzene as having sufficient evidence for carcinogenicity in humans, and that that PCE is probably carcinogenic to humans. IARC noted that the bladder “may be [a] target tissue[] for tetrachlorethylene-induced carcinogenesis in humans...”³² EPA concluded that “TCE is carcinogenic to humans by all routes of exposure,” that is, by ingestion, inhalation, and dermal exposure.³³ Further, EPA concluded that PCE is “likely to be carcinogenic in humans by all routes of exposure.”³⁴ Similarly, the National Toxicology Program has recognized TCE as “known to be a human carcinogen”³⁵ and PCE as “reasonably anticipated to be a human carcinogen.”³⁶ ATSDR’s 2017 Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases found sufficient evidence exists for PCE causing bladder cancer, stating that “the epidemiological studies provide sufficient evidence for causation and are consistent with the mechanistic information that certain genetic polymorphism may enhance the production of genotoxic PCE metabolites in the bladder via the GSH conjugate pathway.” While ATSDR did not find sufficient evidence for TCE and bladder cancer, later studies have strengthened the association as noted by Dr. Hatten. As reported by Dr. Hatten and Dr. Plunkett, epidemiological studies have identified elevated bladder cancer diagnoses associated with benzene and vinyl chloride.³⁷

As reported by Dr. Hatten, Dr. Plunkett, Dr. Gilbert, and Dr. Bird, both TCE and PCE share similar metabolic pathways: toxic metabolites are eventually excreted from the kidneys into urine where it sits in the bladder until voided.³⁸ Dr. Plunkett identifies the same endpoint for benzene and vinyl chloride metabolites as well.³⁹ This means that the toxic metabolites can spend hours in contact with urothelial cells inside the bladder. Below is a figure from Dr. Gilbert explaining the metabolic pathways and outcome for TCE and PCE-induced bladder cancer.

³² International Agency for Research on Cancer. Trichloroethylene, Tetrachloroethylene, and Some Other Chlorinated Agents. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. 2014;106:1-514

³³ Environmental Protection Agency. Toxicological Review of Trichloroethylene (CAS No. 79-01-6). 2011

³⁴ Environmental Protection Agency. Toxicological Review of Tetrachloroethylene (CAS No. 127-18-4). 2012

³⁵ National Toxicology Program (NTP). 2015. Report on Carcinogens monograph on trichloroethylene. Research Triangle Park, NC: National Toxicology Program. RoC Monograph 05

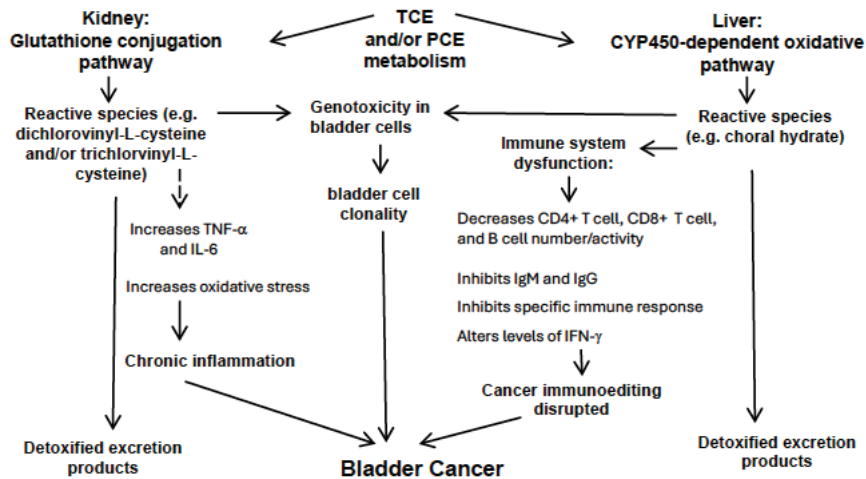
³⁶ NTP (National Toxicology Program). 2021. Report on Carcinogens, Fifteenth Edition. Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service

³⁷ Hatten pp. 26-29; 31-32; Plunkett ¶ 47

³⁸ Plunkett ¶¶ 33, 43; Hatten p. 39; Bird pp. 17-18

³⁹ Plunkett ¶¶ 52, 56, 59

Figure 1. Model for TCE and/or PCE-induced bladder cancer



Dr. Gilbert reports that inhalation and dermal exposure from TCE-contaminated water at least doubles ingestion consumption figures (and with similar evidence for PCE).⁴⁰ Dr. Gilbert further explains that a mixture of TCE, PCE, and benzene can produce additive effects that can cause bladder cancer in that both TCE and PCE share a similar metabolic pathway and all three chemicals promote chronic inflammation and immunosuppression.⁴¹ Regarding chronic inflammation in particular, Dr. Gilbert concludes that it “is an important driver of bladder cancer and provides support for tumor progression, metastasis, and anti-cancer resistance.” In addition, TCE and PCE’s can reduce the impact of the body’s natural immune response to bladder cancer, which is important given that the most common intravesical treatment used to fight bladder cancer – BCG – essentially activates an adaptive immune response.⁴²

Over time, the scientific consensus has progressed to greater certainty, and action, regarding the toxicity of the chemicals at Camp Lejeune. In December 2024 EPA finalized a rule banning on TCE and most commercial uses of PCE under the Toxic Substances Control Act, describing TCE as “extremely toxic” and PCE as “cancer-causing”. As noted by Dr. Bird in his supplemental report, “the EPA determined that any *lesser* restrictions on the use of TCE or PCE would fail to adequately protect public health.”⁴³ Dr. Bird further explained that EPA’s safety measures were based on the wastewater concentrations, not consumption, meaning that the risk for those at Camp Lejeune (whose ingested concentrations alone are than the concentrations identified in the EPA rule) is even greater.

⁴⁰ Gilbert p. 30

⁴¹ Gilbert p. 32-3

⁴² Gilbert p. 19-20

⁴³ Bird Suppl. p. 1

Accordingly, there is a sufficient basis to conclude that the chemicals in the water at Camp Lejeune are capable of causing bladder cancer.

VIII. Differential etiology

In order to assess whether Mr. Criswell's exposure to the water at Camp Lejeune as likely as not caused him to develop bladder cancer, I employed a differential etiology under which an expert considers the relevant potential causes of a disease and then attempts to ascertain whether any of those causes can be eliminated. I employ this analysis in my practice to assist in the treatment options I provide my patients.

During an encounter for bladder cancer, a treating physician will often try to establish a differential etiology for the development of the patient's disease. (An important distinction, this is a differential etiology, not a diagnosis. The diagnosis is bladder cancer, the cause is the etiology.) This is a thorough, but not exhaustive line of questioning, because the cause of the disease is of less importance than the therapy plan of the disease for the treating physician. The most immediate goal of the visit for a treating physician is to develop the appropriate treatment plan for the patient. However, in a disease like bladder cancer, whose etiology is almost always from an exposure, it is worthwhile to identify the exposure. This will make the patient aware and permit them to avoid the exposure going forward if it is possible. It may also afford an opportunity to warn others, particularly family and coworkers, with similar exposures. Gathering this data also reveals patterns that may be important to the health of society as a whole. In fact, this revelation of patterns leads to the science of epidemiology discussed above.

It is not uncommon for bladder cancer to develop in a patient with more than one risk factor for the development of bladder cancer. Behavior based, environmental, and occupational exposures are well-established risk factors for the development of bladder cancer, and the American Cancer Society recognizes that multiple exposures – such as smoking and workplace exposures – “can act together to cause bladder cancer.”⁴⁴ These risk factors act in concert with one another, and have additive effects. In Mr. Criswell's case, as I discuss later in this report, he only had one recognized risk factor: exposure to the chemicals in the water at Camp Lejeune.

Before addressing the potential risk factors for bladder cancer, it is important to address whether the cause of bladder cancer is idiopathic. In a deposition of Mr. Criswell's treatment provider – Dr. Shelfo – the government asked whether the cause of bladder cancer is idiopathic.⁴⁵ When a disease is idiopathic it means that the cause of the disease is unknown. In my opinion, bladder cancer is rarely idiopathic in the sense that it is likely to have a known cause. In situations where an individual was exposed to a known cause of bladder cancer, considering whether the cause is idiopathic is inapplicable. As I

⁴⁴ <https://www.cancer.org/content/dam/CRC/PDF/Public/8558.00.pdf>

⁴⁵ Shelfo Dep. 33:4-12